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LETTER TO THE EDITOR

The equine Hendra virus vaccine remains a highly effective preventative measure against infection in horses and humans: 'The imperative to develop a human vaccine for the Hendra virus in Australia'

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To the Editor

In their commentary article, 'The imperative to develop a human vaccine for the Hendra virus in Australia',

Zahoor and Mudie (1) argue the case for a human Hendra virus (HeV) vaccine. The statements supporting their arguments are incorrect and have the potential to cause

confusion and ultimately undermine confidence in current evidence-based risk management strategies, thereby placing equine and human lives at risk.

The central argument in Zahoor and Mudie (1) is that HeV is 'rapidly mutating', with consequent loss of efficacy of the equine HeV vaccine, changing clinical syndromes in humans, and infection in new animal species. There is no scientific basis to their central argument. Zahoor and Mudie (1) offer no citations to support their statements regarding the mutation rate of HeV. Indeed, primary research indicates the HeV genome has minimal variability (less than 1% at both the nucleotide and amino acid levels) in both flying-foxes and horses and is highly stable (the same variant has been detected at disparate locations at the same time, and over periods of at least 12 years) (2, 3).

There is no evidence that the equine HeV vaccine is becoming less effective. Continuing equine HeV cases do not reflect loss of vaccine efficacy as stated by Zahoor and Mudie (1), but rather a failure of some horse owners to vaccinate their horses. There have been no HeV cases in vaccinated horses. The efficacy and safety of the recombinant equine vaccine has been clearly demonstrated (4–6), and both government and industry animal health authorities strongly recommend its use as 'the single most effective way of reducing the risk of Hendra virus infection in horses' (7).

There is no evidence that the nature of human HeV infection is changing. The seven recognised human cases have shared clinical features but are insufficient in number to determine changes over time (8–13).

There is no evidence that recently reported canine cases indicate that HeV is 'seeking new co-hosts'. The wide host range of HeV in experimental studies is well established (14, 15). The two observed cases of natural HeV infection in dogs most likely resulted from exposure to infected horses, or contaminated material from these horses, and their detection may reflect increased surveillance of canines on infected equine premises (16).

There is no evidence that HeV infections 'are emerging in locations far beyond bats' typical migratory boundaries'. Several recent publications demonstrate that the spatial occurrence of equine HeV cases reflects the distribution of black and spectacled flying-foxes (17–19).

In conclusion, we express no objection to the development of a human vaccine against HeV; however, we are emphatic that Zahoor and Mudie (1) are unjustified in using viral evolution, vaccine inefficiency, and changing clinical syndromes as motivations. There are no data to support their case.

Conflict of interest and funding

Dr. Broder reports a grant (CRADA) from Zoetis, Inc., outside the submitted work. In addition, Dr. Broder is a coinventor on U.S. Patent No. 8,865,171 and 9,045,532, with royalties paid by Zoetis, Inc., and Australian Patent

No. 2005327194 Patent assignees are the United States of America as represented by the Department of Health and Human Services (Washington DC) and the Henry M. Jackson Foundation (Bethesda, MD).

Dr. Restif reports grants from The Royal Society, during the conduct of the study; he further discloses that he is the sponsor of a Junior Research Fellowship supported by the Axa Research Fund.

Dr. Dhand has communicated with Zoetis for submitting a joint ARC linkage project but this submission did not materialize.

Dr. Secombe is a core executive member of Equine Veterinarians Australia.

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